WE CLAIM:

1. A method of diagnosing colorectal cancer or hyperplastic polyposis in a subject, said method comprising determining the degree of methylation of nucleic acid that regulates expression of an *MCC* gene in a sample derived from the subject, wherein an enhanced degree of methylation in the sample relative to a suitable control sample is indicative of colorectal cancer or hyperplastic polyposis.

2. A method for determining a predisposition for neoplastic transformation in a subject having colorectal lesions or polyps said method comprising determining the degree of methylation of nucleic acid that regulates expression of an *MCC* gene in a sample derived from a lesion or polyp wherein an enhanced degree of methylation in the sample relative to a suitable control sample is indicative of a predisposition for neoplastic transformation in the subject.

3. A method of monitoring the efficacy of treatment for colorectal cancer or hyperplastic polyposis in a subject, said method comprising treating a subject in need of treatment for colorectal cancer or hyperplastic polyposis and determining the degree of methylation of nucleic acid that regulates expression of an *MCC* gene in a sample derived from the subject, wherein a degree of methylation in the sample comparable to that for a suitable control sample is indicative of effective treatment.

4. A method of monitoring the efficacy of treatment for colorectal cancer or hyperplastic polyposis in a subject, said method comprising treating a subject in need of treatment for colorectal cancer or hyperplastic polyposis and determining the degree of methylation of nucleic acid that regulates expression of an *MCC* gene in a sample derived from the subject, wherein a hyper methylation of the *MCC* promoter in the sample compared to the level of methylation for a suitable control sample indicates that treatment is not effective.

5. The method according to any one of claims 1 to 4 wherein the colorectal cancer is a carcinoma *in situ*.

6. The method of claim 5 wherein the carcinoma *in situ* is characterized by the presence of polyps in the proximal and distal colorectum.

7. The method of claim 6 wherein the polyps are hyperplastic polyps.

8. The method according to any one of claims 1 to 4 wherein the colorectal cancer is a stage A colorectal cancer.

9. The method according to any one of claims 1 to 4 wherein the colorectal cancer is a stage B colorectal cancer.

10. The method according to any one of claims 1 to 4 wherein the colorectal cancer is a stage C colorectal cancer.

11. The method according to any one of claims 1 to 4 wherein the colorectal cancer is a stage D colorectal cancer.

12. The method according to any one of claims 1 to 11 wherein the sample comprises tissue or cells from the colorectum, colorectal polyps, or a stool sample.

13. The method according to any one of claims 1 to 12 wherein determining the degree of methylation of nucleic acid that regulates expression of an *MCC* gene in a sample consists of determining the degree of methylation in a region of an *MCC* gene consisting of nucleotides 1 to 560 of SEQ ID NO: 3 or a complementary sequence thereto.

14. The method according to any one of claims 1 to 12 wherein determining the degree of methylation of nucleic acid that regulates expression of an *MCC* gene in a sample consists of determining the degree of methylation in a region of an *MCC* gene

consisting of nucleotides 292 to 458 of SEQ ID NO: 3 or a complementary sequence thereto.

15. The method according to any one of claims 1 to 12 wherein determining the degree of methylation of nucleic acid that regulates expression of an *MCC* gene in a sample consists of determining the degree of methylation in a region of an *MCC* gene consisting of nucleotides 284 to 304 of SEQ ID NO: 3 or a complementary sequence thereto.

16. The method according to any one of claims 1 to 12 wherein determining the degree of methylation of nucleic acid that regulates expression of an *MCC* gene in a sample consists of determining the degree of methylation in a region of an *MCC* gene consisting of nucleotides 335 to 355 of SEQ ID NO: 3 or a complementary sequence thereto.

17. The method according to any one of claims 1 to 12 wherein determining the degree of methylation of nucleic acid that regulates expression of an *MCC* gene in a sample consists of determining the degree of methylation in a region of an *MCC* gene consisting of nucleotides 361 to 404 of SEQ ID NO: 3 or a complementary sequence thereto.

18. The method according to any one of claims 1 to 17 comprising performing methylation-sensitive endonuclease digestion of DNA from the sample.

19. The method according to any one of claims 1 to 17 comprising hybridizing a probe comprising an *MspI*/HpaII recognition site to nucleic acid digested with *Hpa*II enzyme for a time and under conditions sufficient to digest non-methylated DNA.

20. The method of claim 19 wherein the probe comprises nucleotides from about position 291 to about position 294 of SEQ ID NO: 3 or a complementary sequence thereto.

21. The method according to any one of claims 1 to 17 comprising subjecting nucleic acid from the sample to DNase I digestion and amplifying the fragments produced using primers that bind to or flank methylated regions in the *MCC* gene promoter comprising a sequence from position 1 to about position 560 of SEQ ID NO: 3 or a complementary sequence thereto.

22. The method of claim 21 wherein the primers flank a region of the *MCC* gene including residues 284-404 of SEQ ID NO: 3 or a complementary sequence thereto.

23. The method of claim 21 or 22 wherein a primer comprises a nucleotide sequence selected from the group consisting of SEQ ID NOs: 11-16 or 18-23 or 25-34 or a complementary sequence thereto.

24. The method according to any one of claims 21 to 23 further comprising detecting the amplified fragments with a nucleic acid probe capable of specifically hybridizing to the amplified fragment.

25. The method of claim 24 wherein the probe comprises a sequence set forth in SEQ ID NO: 12 or 15 labelled at its 5-end and 3-end with one or more fluorescent ligands and/or a Black Hole Quencher (BHQ).

26. The method of claim 25 wherein the fluorescent ligands are slected from the group consisting of TAMRA and FAM.

27. The method according to any one of claims 1 to 17 comprising treating nucleic acid from the sample with an amount of a compound that selectively mutates non-methylated cytosine residues in nucleic acid under conditions sufficient to induce mutagenesis.

28. The method of claim 27 wherein the compound is a metal salt of bisulphite.

29. The method of claim 28 wherein the compound is sodium bisulphite or potassium bisulphite.

30. The method according to any one of claims 27 to 29 further comprising amplifying a fragment using primers that flank a methylated cytosine residue or mutated residue at an equivalent position in non-methylated nucleic acid.

31. The method of claim 30 wherein the primers flank a region of the *MCC* gene including residues 284-404 of SEQ ID NO: 3 or a complementary sequence thereto.

32. The method of claim 30 or 31 wherein a primer comprises a nucleotide sequence selected from the group consisting of SEQ ID NOs: 11-16 or 18-23 or 25-34 or a complementary sequence thereto.

33. The method according to any one of claims 30 to 32 further comprising detecting the amplified fragments with a nucleic acid probe capable of specifically hybridizing to the amplified fragment.

34. The method of claim 33 wherein the probe comprises a sequence set forth in SEQ ID NO: 12 or 15 labelled at its 5 -end and 3 -end with one or more fluorescent ligands and/or a Black Hole Quencher (BHQ).

35. The method of claim 34 wherein the fluorescent ligands are selected from the group consisting of TAMRA and FAM.

36. The method according to any one of claims 1 to 35 wherein the enhanced degree of methylation consists of at least about 3 CpG islands in the nucleic acid being methylated in the sample relative to a suitable control sample.

37. The method according to any one of claims 1 to 36 further comprising determining the degree of methylation of a control sample.

38. The method according to any one of claims 1 to 37 wherein a control sample is a data set selected from the group consisting of:

a) a data set comprising measurements of the degree of methylation for a typical population of subjects known to have colorectal cancer;

b) a data set comprising measurements of the degree of methylation for the subject being tested wherein said measurements have been made previously, such as, for example, when the subject was known to healthy or, in the case of a subject having colorectal cancer, when the subject was diagnosed or at an earlier stage in disease progression;

c) a data set comprising measurements of the degree of methylation for a healthy individual or a population of healthy individuals;

d) a data set comprising measurements of the degree of methylation for a normal individual or a population of normal individuals; and

e) a data set comprising measurements of the degree of methylation from the subject being tested wherein the measurements are determined in a matched sample.

39. The method according to any one of claims 1 to 38 further comprising isolating the sample from the subject.

40. The method of claim 39 wherein the sample is derived by colonoscopy.

41. The method according to any one of claims 1 to 41 further comprising determining the level of methylation another gene selected from the group consisting of *HLTF, APC, p16^{INK4a},p14^{ARF}, HPP1, hMLH1, MGMT,* and combinations thereof wherein hypermethylation of *MCC* and the other gene is indicative of colorectal cancer or hyperplastic polyposis or a predisposition of a lesion or polyp to neoplastic transformation.

42. The method according to any one of claims 1 to 41 further comprising determining a CpG island methylation phenotype (CIMP) for the subject wherein methylation of at least about 3-4 CIMP markers in combination with hypermethylation of *MCC* is indicative of colorectal cancer or hyperplastic polyposis or a predisposition of a lesion or polyp to neoplastic transformation.

43. The method according to any one of claims 1 to 41 further comprising determining a mutation in the *APC* gene or *DCC* gene wherein hypermethylation of *MCC* in combination with a deletion or point mutation of *APC* or *DCC* are indicative of of colorectal cancer or hyperplastic polyposis or a predisposition of a lesion or polyp to neoplastic transformation.

44. An isolated nucleic acid probe or primer that is capable of selectively hybridising to a region of the *MCC* gene promoter that is hyper methylated in a colorectal cancer, wherein said region comprises or is contained within nucleotide residues from about position 284 to about position 404 of SEQ ID NO: 3 or SEQ ID NO: 17 or SEQ ID NO: 24 or a complementary sequence thereto.

45. A probe or primer consisting of a nucleotide sequence set forth in any one SEQ ID NOs: 11-16 or 18-23 or 25-34 or a complementary sequence thereto.